

FAQ for Muscle Physiology Lecture given by Dr. Abraham D. Lee

I collected questions I received and provided answers below. I hope that it is helpful to you.

Q: My first question was about the Type I and Type II fibers. I know we can't change from Type II to Type I with distance training, but you said that distance runners can change from Type IIb to Type IIx to Type IIa, but earlier you said that Type IIb is only in rats. So was that not actually an endurance test of muscle Type in humans, just in rats?

A: I am sorry that I was not clear about it. Yes, in humans endurance training will transform type IIx into type IIa and in rodents i.e. rats, type IIb into type IIx, which then into type IIa

Q: Should we just memorize the exceptions to the "General Rule" we discussed in class?

A: Understand the general rule and exceptions

Q: What were examples of concentric actions vs. eccentric action? Are they typically correlated with each other (like, with biceps and triceps, if one is doing concentric action, the other is doing eccentric action)?

A: If you hold a dumbbell and flex elbow with the elbow fixed at the side, the biceps is doing concentric contraction. If one muscle is doing one action (concentric) and other opposite muscle is usually passive. Sometimes agonists and antagonists do co-contractions concomitantly.

Q: I was studying the lecture from today and on pg. 228 I had a question about the muscle fiber composition. I believe you said the difference between the type I and type II muscle fiber composition in sprinters and long distance runners is due to genetic differences. I understand that there is no transformation between type I and type II. But then why would there be a difference between the muscle fiber compositions in individuals with spinal cord injury? Why would there be a decrease in type I fibers if there is no genetic difference?

A: Your question is very good. According to data available in literature, there is no transformation between type I and type II with EXERCISE TRAINING.

An increase in type II fibers in an individual with spinal cord injury is due to different internal stimuli, which is denervation. Denervation is different from Exercise or Non-Exercise. I do not know mechanisms underlying fiber type change with SCI. I may look for research articles. If you come across research articles, let me know. I hope I am answering your question. Let me know if I have helped you. Good luck on the exam on Monday! Thank you.

Q: On the slide "Comparison b/n non-pennated & pennated muscle," it indicates that the "muscle force production" for pennated is "greater" and for non-pennated is "less." However, I thought based on the previous slide before this one, that the force of pennation was only 87%, i.e., that there was some loss in force due to pennation.

A: At the level of muscle fibers, it is true that pennated fiber lose its force due its pennation. But at the muscle level, muscle that has pennated fibers has more fibers than

muscle that have nonpennated fibers. Thus, muscle, which consists of pennated fibers, can produce more force than does muscle, which consists of nonpennated fibers.

Q: What is the difference between muscle torque and muscle power? Are these terms interchangeable?

A: Muscle torque is the product of force X force arm distance. It is usually expressed in N.m. In my lecture, I used muscle torque and muscle force interchangeably. Precisely speaking it is incorrect.

Power is the product of force x speed. It is usually expressed in N.m/sec, which is equivalent to Watts.

Q: Also, could you elaborate on the relationship between intensity and fiber type?

A: Your third question is related to motor unit recruitment. If you do exercise at a low intensity, you will recruit slow oxidative motor unit. As you increase intensity of exercise, you will gradually recruit FR MU and then eventually FF MU at high intensity. Let me know if I have helped you. If you need more help, let me know. Thank you.

Q: Thank you for such a good lecture today! Could you please give me example of an isometric contraction?

Here are examples for isometric contractions:

- 1) Pushing against wall or table with your arms
- 2) Keeping a dumbbell (i.e., 5-10 lbs) in your hand with elbow joint at 90 degree flexion. In this condition your biceps work isometrically.

Q: What does SC mean on page 252?

A: SC means spinal cord.

Q: In slide #5, Effect of Pennation, it shows that pennation results in force loss (.87x) and also space saving. However, in slide #6, Comparison b/n non-pennated & pennated muscle, the table shows that pennated does have force loss due to pennation, but also shows that muscle force production is greater in pennated than in non-pennated muscles.

A: It is because pennated muscle has more muscle fibers than does non-pennated muscles. We are not comparing force production between a pennated fiber and a nonpennated fiber but comparing between muscle that has pennated fibers and muscle that has non-pennated fibers.

Q: Also, the table shows that pennated has greater cross sectional area, but if it is involved in space saving, why does it have a greater cross sectional area than non-pennated muscles?

A: Here the cross sectional area refers to the physiological cross sectional area (PCSA) of muscle. PCSA is a sum of all individual fiber area within a muscle of interest. Furthermore, each fiber area is perpendicular to the longitudinal axis of each fiber. That is, the more the fibers are present in a muscle, the larger the PCSA of the muscle will be. Therefore, usually a muscle that consists of pennated fibers will have a greater PCSA than does a muscle that consists of non-pennated fibers.

Q: I have one brief question regarding today's muscle lecture. Can you please explain why spinal chord injuries result in an increase in the percentage of type II fibers (and an associated decrease in the percentage of type I fibers)?

A: That is an excellent question. At this point, I do not know the answer. I did not have chance to come across any research paper that explains mechanisms underlying SCI-induced changes in muscle fiber transformation. It definitely related to the effect of denervation due to SCI. Let me know if you see a research article, which explains these mechanisms.

Q: Do we have to know the list of names of all the flexors and extensors of the body that were on the slide you presented on yesterday?

A: Yes, it is better to know them, only those covered during the class. Thank you.

Q: Is it necessary to memorize all of the examples of flexors and extensors on pg. 237 of the notes?

A: Yes.

Q: Do Type IIx fibers utilize aerobic and anaerobic respiration or simply anaerobic? You said it was in between type IIa and IIb but I wanted to clarify that point.

A: There is no muscle fiber which utilize only one energy system. All muscle fibers utilize both aerobic and anaerobic metabolic pathway to derive ATP. But type I fibers depend predominantly on aerobic system. On the other hand, type IIb fibers depend predominantly on anaerobic system. Type IIa fibers have capability to utilize both aerobic and anaerobic metabolic pathways to derive energy. It could be ~50% from aerobic and ~50% anaerobic energy systems, depending on the intensity of exercise.

Type IIx fibers also possess aerobic capacity, which is less than that in the type IIa but greater than that in type IIb. They also possess anaerobic capacity, which is greater than that in the type IIa but less than that in type IIb.

Q: In Class, you were explaining how different degrees of muscle positions generated certain levels of force. I was wondering if it was necessary to know which degree (0 or 45 degrees) generate more force for specific muscle groups (Flexors, Extensors, Etc.).

A: Probably I may not ask you how much muscle force is produced at a certain degree by a certain muscle. But the important thing that you need to know is the pattern of muscle force production by a certain muscle throughout the range of motion during flexion and/or extension.

Q: Anterior deltoid is considered a flexor in that it is a muscle that causes two segments to move close to each other within in a sagittal plane. What are the two segments that move closer together?

A: This is another good question. Intuitively, it is not clear about the two segments that move close together during the action of shoulder flexion by anterior deltoid.

I would like to suggest you to think of an imaginary vertical line which extends from your shoulder. Also assume that this imaginary line does not move during shoulder joint flexion. Then you can see your upper arm and this imaginary line move close together during the shoulder flexion by the anterior deltoid. I hope this may help you.

Q: On the slide labeled "Characteristics of muscle fibers", it talks about "ATPase activity". Is this referring to myosin ATPase or Ca²⁺ ATPase?

A: It refers to myosin ATPase.

Q: Why do the type II muscle fiber fatigue faster than does type I muscle fiber?

A: This is related to energy source muscle fibers depend on during contraction and the number of mitochondria present in muscle fibers. Type 2 muscle fibers depend heavily on anaerobic energy source such as glycogen, ATP, and phosphocreatine (PCr). ATP and PCr are limited and used up quickly. Glycogen lasts longer since it is stored in greater amount than ATP & PCr. But the byproduct of the breakdown of glycogen is lactic acid, which disturbs intracellular pH, making it acidic. This is a part of causing fatigue. On the other hand, type I fibers rely on aerobic energy source through oxidative phosphorylation in mitochondria. Typically fat such as triacylglycerol is used in mitochondria for energy production without any accumulation of metabolites, which may cause the fiber fatigue.

Q: Why do type II fibers generate peak force quickly and recover from the peak force quickly than do type I fibers?

A: Quicker peak force production and quicker recovery by type II muscle fibers compared with type I fibers is associated with quicker Ca²⁺ release from SR and quicker uptake of Ca²⁺ into SR. Also remember that the activity of myosin ATPase in type II fibers is faster than that in type I fibers. This is another reason why type II fibers generate peak force quicker and recover quickly than do type I fibers.

Q: In the Force-Velocity Curve for muscle it shows that eccentric contraction can generate larger maximum force than concentric contraction. Is this always true?

A: Yes

Q: Do muscles always generate more force during eccentric action than concentric action no matter what the speed of contraction is?

A: Yes

Q: Also, this graph makes it seem like the lengthening action of muscles generates the same amount of force no matter what the speed of contraction is. Is that true?

A: Yes, it is true. During eccentric contraction, the force (torque) production of muscles is almost independent of the speed of movement. This is not the case during concentric contraction.

Q: We are told that there is NEVER conversion between muscle types (i.e. Type IIb → Type IIx). Does this mean that cells can actually change the *number* of each type of Type II fibers they have?

A: Actually there is a conversion between type II muscle fibers with exercise training. In human muscles there is conversion from type IIx to type IIa with both endurance and resistance trainings. But it is not accepted in general that type II is converted into type I fibers in humans. Hyperplasia (an increase in # of muscle fibers) is minimal if at all in humans with exercise training.

Q: On the force - velocity curve, why does the force produced by lengthening action of the muscle not change at all with varying velocity?

I do not know an answer for the question above. This is an observed result. That means that there is no disadvantage or advantage in either mechanical or biochemical mechanism in producing force during eccentric action of muscle. This is in contrast to a disadvantage observed during shortening action with an increase speed, which might be due to lack of full interaction between myosin head and action filaments during fast contraction.

Q: On the force vs knee flexion degree graph, why does the curve follow the general rule for an extended hip, but with a flexed hip the force generated by the knee flexor muscle increases as the angle increases? Isn't the muscle still shortening (and therefore decreasing force output)? What does the hip have to do with this?

First of all, I like to remind you that you are responsible for knowing **the graph only with hip extended** not the graph with hip flexed.

To answer your questions:

- 1) Hip flexion causes the lengthening of knee flexors compared with 0 degree hip flexion. I hope that you can visualize this by lying on stomach with one pillow in front of one of your hip joints.
- 2) With hip in a flexed position, which will lengthen knee flexors, slight knee flexion (20-40 degrees) may favor of having biomechanical advantage such as force arm distance compared with that with 0 degree or less than 20 degree flexion in spite of muscle shortening. But eventually further increase in knee flexion causes a decrease in force produced by knee flexors per the general rule.